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Probable Evidence of Scurvy in Subadults From Archeological Sites in Peru

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ABSTRACT Subadult scurvy is not well documented in archeological human remains despite the existence of many biomedical references indicating that bone changes do occur in some cases and, because of this, should be observable in human burials. There are several potential reasons for this gap in our knowledge of scurvy. Not all children who suffered from scurvy died of the disease or from other causes when they had scurvy. Scurvy may not leave characteristic bone changes in every case of the disease. Some of the pathological conditions associated with scurvy have been known for many years, but these features may be rare or difficult to differentiate from other pathological conditions. Recently a lesion of the skull has been described (Ortner and Ericksen [1997] International Journal of Osteoarchaeology *7*:212–220) that is probably pathognomonic for scurvy, specifically porous and sometimes hypertrophic lesions of the greater wing of the sphenoid. This lesion is bilateral and highly associated with evidence of inflammation at other anatomical sites in the skull. A survey of subadult skulls (N = 363) in the human skeletal collection from Peru at the National Museum of Natural History, Smithsonian Institution, reveals a prevalence of 10% of skulls that exhibit plausible evidence of scurvy. Some cases of scurvy also have cribra orbitalia that has been attributed to anemia. In most of the Peruvian scurvy cases, anemia is an unlikely possibility because there is no evidence of marrow hyperplasia. This highlights the need for caution in using lesions of the orbit as an indicator of anemia when there is no other evidence of this disease elsewhere in the skeleton. Anatomical evidence of scurvy offers the potential of providing new and important evidence of diet in archeological human populations. Am J Phys Anthropol 108:321–331, 1999.

Three diseases that result from a nutritional deficiency have been identified in archeological human skeletal remains: 1) rickets or vitamin D deficiency (Ortner and Mays, 1998), 2) scurvy or vitamin C deficiency (Ortner and Ericksen, 1997), and 3)

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iron deficiency anemia (Von Endt and Ortner, 1982; Stuart-Macadam 1987, 1989). Because of the underlying relationship to malnutrition, two or three of these diseases may occur in the same group of people and are known to occur clinically in the same patient (Follis et al., 1940; Seaman and Rivers, 1989; Vilter, 1964). Since the prevalence of each of these diseases provides important data on diet in archeological human populations, careful attention is necessary to define the pathological features that are associated with each disease. Ideally, we need to be able to differentiate among the three conditions as well as between any of the three and other pathology. This will not always be possible, but periodic reevaluation and revision of relevant criteria will improve diagnostic accuracy.

The pathological feature central to the lesions of bone apparent in scurvy is an area of abnormal porosity of the cortex. This increased porosity may be accompanied by bone hyperplasia; however, this is much less common than porosity alone. The biological mechanism of this pathological phenomenon is a vascular response to chronic bleeding at the site of the porosity/hyperplasia. Abnormal bleeding seen as hemorrhages in various tissues is a well-established clinical feature of scurvy (Jaffe, 1972). A common hypothesis on the pathogenesis of hemorrhaging in scurvy is that the intercellular cement material of blood vessels formed during a scorbutic episode is deficient and weak, and this deficiency results in bleeding from minor trauma (Jaffe, 1972). This mechanism has been challenged by Hodges (1980), but clearly abnormal bleeding and hemorrhage do commonly occur in scorbutic patients.

Extravasated blood, particularly if chronic, evokes a vascular inflammatory response that includes increased blood vessels at the site. Some of the increased vascularity will occur as blood vessels penetrate the underlying cortex, creating the holes seen as porosity in bone tissue. The periosteum may also be stimulated by bleeding between the cortex and periosteum (Ortner and Putschar, 1985). When this happens in infants and children, the loosely attached periosteum is stripped from the underlying bone, which activates bone formation.

TABLE 1. Sites, archeological dates, and number of probable cases of scurvy in subadult crania from Peru

Site	Archeological date	Cases of scurvy	Total subadults
Lima, site?	Before 1530 AD	2	6
Junin, Caudivilla	1100–1400 AD	1	3
Lima, Huarochiri	1200–1500 AD	1	12
Libertad, Chicama	300–800 AD	8	89
Lima, Pachacamac	500–1500 AD	19	149
Lima, Chilca	2000 BC–300 AD	2	18
Lima, San Damian	?	2	16
Lima, Huacho	500–1450 AD	2	6
Lima, Chillon Valley	400–1300 AD	1	1
Other sites	Various dates	0	63
Total		38	363

On the basis of anatomical features associated with scurvy as reported by Ortner and Ericksen (1997), the authors of this report initiated a survey of all the subadults (N = 363) in the Peruvian human skeletal collection of the National Museum of Natural History, Smithsonian Institution. The lesion that in our opinion is most pathognomonic for scurvy is the porous lesion of the greater wing of the sphenoid. Our objective was to identify all cases having this lesion and to determine if there were other pathological features in the skull that were associated with the sphenoid lesions.

MATERIALS AND METHODS

The skeletal samples used in this research are from many archeological sites in Peru. Most of the sites are in the coastal lowlands, but two are located in the higher elevations adjacent to the coastal plain (Junin, Caudivilla, and Lima, Huarochiri, in Table 1). Sites that contained subadults but no evidence of scurvy (Other sites in Table 1) were also mostly from coastal lowland sites, but there also were a few skulls from the highlands. Most of the human remains were collected by Dr. Aleš Hrdlička during expeditions to Peru in the early part of this century. All the remains are now curated and stored at the National Museum of Natural History,

Fig. 1. Abnormal porosity of the greater wing of the sphenoid, frontal and maxilla in the skull of a child 18–24 months of age. NMNH 293908 from the site of Pachacamac, Peru.

Fig. 2. Detail of porous cortical bone of the greater wing of the sphenoid in Fig. 1.

Smithsonian Institution. The archeological provenance was not carefully established at the time of excavation, so associated archeological dates are imprecise at best.

The adverse effects of scurvy are most likely to be manifest in individuals undergoing active growth. For this reason, we limited our analysis to subadults defined on the basis of either or both of two age-related features: 1) complete eruption of the third molar and 2) fusion of the basi-occipital synchondrosis. If the third molar was at the occlusal plane or the basi-occipital synchondrosis was fused, the skull was not included in the sample. Making this decision was complicated at times by abnormalities in the eruption of the third molar or postmortem breakage of the skull base. Subadult skulls in which the relevant anatomical sites were missing were excluded from the sample. Mandibles were rarely present, and no postcranial bones associated with the skull were available for analysis.

Each of the skulls was evaluated for evidence of cortical pathology on the greater wing of the sphenoid (Figs. 1, 2). The primary pathology was abnormal porosity at this site, although in one case the porosity was accompanied by bone hypertrophy. Porosity is defined as a localized, abnormal condition in which fine holes, visible without or with low magnification and typically less than 1 mm in diameter, penetrate a lamellar bone surface. In rare situations, the increased porosity is accompanied by bone hypertrophy. Porous bone as we use it is the result of chronic inflammation and needs to be distinguished from porotic hyperostosis resulting from hyperplasia of hematopoietic marrow (Ortner and Ericksen, 1997). In our use of the term *inflammation*, we follow the discussion of this subject by Janeway and Travers (1994) and imply only an abnormal increase in vascularity and the presence of inflammatory cells associated with the removal of extravasated blood. We do not imply the presence of other inflammatory cells such as lymphocytes.

In all cases exhibiting abnormal porosity of the greater wing of the sphenoid, we evaluated other sites on the skull for evidence of inflammatory bone changes (Table 2). Lesions were recorded as absent, present, or nonobservable. The location on the left and/or right side was recorded. In Table 2 we have listed alveolar processes and sockets as sites where inflammatory features will occur in scurvy. Abnormality at these sites undoubtedly occurs (Fig. 3), but in subadult skulls some porosity is a normal part of dental eruption and growth. We were unable to establish a normal baseline for porosity of these sites and did not include these features in our research. However, in future research the range of normal and abnormal

TABLE 2. Anatomical sites associated with scorbutic lesions in subadult skulls

Cranium	Mandible
Cranial vault	Coronoid process, medial surface
Greater wing of the sphenoid	Alveolar processes
Orbit, frontal (roof)	Alveolar sockets
Orbit, zygomatic (lateral)	
Maxilla, posterior surface	
Zygomatic bone, internal surface	
Infraorbital foramen	
Alveolar processes	
Alveolar sockets	
Palate	

Fig. 3. Porous lesions of the left maxilla and alveolar process in the skull of a child 3–5 years of age. NMNH 293169 from the site of Chilca, Peru.

Fig. 4. Woven bone surface of the greater wing of the left sphenoid in an infant about 1 year of age. NMNH 382638 from the site of Quicksburg, Virginia. Note the minimal number of fine, vascular holes penetrating the cortex. This surface probably represents a normal expression of rapid growth in an infant and should not be confused with the porosity associated with scurvy.

expressions of porosity for these bone tissues needs to be established.

RESULTS

All of the purported cases are diagnosed as scorbutic primarily on the basis of pathological, porous lesions of the greater wing of the sphenoid. Vascular channels normally pass through the cortex of the sphenoid. These channels typically are larger and much fewer in number than those encountered in cases of scurvy. Scorbutic porosity of the greater wing of the sphenoid is characterized by more numerous, smaller holes penetrating the cortex. In infants there is an additional complication. Virtually all the bone formed in utero and during the first year postnatal is woven (fiber) bone. This bone is rapidly formed and less well organized than compact bone. Its surface anatomy resembles the early stages of periosteal reactive bone formation seen in various pathological processes and can also appear porous (Fig. 4). Careful inspection with a hand lens or dissecting microscope reveals that the porous-appearing surface is very irregular, but the depressions in the surface rarely penetrate entirely through the cortex.

Scurvy can occur in infants, and it may be that some of the woven bone formed is in fact the result of periosteal stimulation from bleeding. However, this possibility needs further investigation. For purposes of this report, pores must regularly penetrate

TABLE 3. Age distribution of probable cases of scurvy in the subadult skeletal samples from archeological sites in Peru

Age category	Total skulls in age category	Total cases of scurvy	Percent of total within age categories
Birth to 2 years	11	2	18
3–6 years	74	6	8
7–12 years	163	18	11
13–18 years	115	12	10
Total	363	38	

TABLE 4. Bilateral distribution of porous lesions of the greater wing of the sphenoid and orbital roof

	Absent	Bilateral	Unilateral	Not observable
Greater wing of sphenoid	0	35	1	2
Orbital roof	1	36	1	0

through the cortical bone tissue to be considered evidence of scurvy. This eliminated many cases of possible scurvy, so the frequency of infants with this disease may be underrepresented in our sample. However, until the abnormal dimension of woven bone formation can be clarified, this seems a more cautious and appropriate way of defining abnormal porosity in infants. After 1 year of age, much more of the bone formed is compact bone, and the determination of abnormal porosity is much easier.

In Table 3 we provide the age distribution of the total subadult sample and the 38 subadults diagnosed as having scurvy. Since growth is much more rapid in early childhood, the probability of forming defective blood vessels should be greater at this stage of development. Consequently, the likelihood of chronic bleeding resulting in inflammatory lesions of the underlying cortex should be greater and a higher proportion of scorbutic cases should be seen in this age category.

The distributions of both the total sample and the scorbutic subsample are clearly weighted toward the older end of the subadult age range. In most archeological burial contexts, the distribution would be greatest in the birth to 6 year category (e.g., Ubelaker 1981). The distribution of the Peru sample used in this report probably reflects a bias favoring older subadults when the samples were collected. We have tried to control for the variation in the number of individuals associated with each of the age categories by determining the percentage of scorbutic cases in each age category relative to the total number of subadults in that category. In all but the infant category, the percentage of scorbutic skulls is similar. The percentage of scorbutic infants is higher, but this is also the category with the smallest total number of subadults, so this figure may be the result of an inadequate sample. The unexpected finding is that there is minimal difference in the prevalence among the age categories.

The overwhelmingly bilateral nature of lesions of the greater wing of the sphenoid and highly associated lesions of the orbital roof is apparent in Table 4. Of the 36 cases of subadult scurvy where observations could be made on both sides, lesions of the greater wing of the sphenoid were bilateral in 35 individuals (97%). Lesions of the orbital roof (Fig. 5) were present in 37 of the 38 cases of scurvy (97%), and most of these lesions were bilateral (97%).

Additional evidence of inflammation is apparent at other anatomical sites in the skull and mandible (Table 5). Twenty-five cases (66%) had porous lesions somewhere on the cranial vault. The most typical site for these lesions was in the lambdoid region (Fig. 6). Seven cases (18%) showed evidence of abnormal bone formation (hyperostosis). All of the cranial vault lesions were relatively slight, and none were associated with hyperplasia of the diploë.

The orbital roof included both porous and hypertrophic lesions. Porosity was very common (97%) in skulls with porous lesions of the greater wing of the sphenoid. Hyperostosis was less common (61%) but was still a frequent condition. Both types of orbital lesions may be associated with other diseases including anemia and infection, and differentiating between causes requires evidence of disease in other areas of the skeleton. It is the association of the orbital roof lesions to the other lesions on the skull also caused by inflammation that provides a pattern of pathology which argues for a diagnosis of scurvy. On the zygomatic (lateral) portion of the orbit, porous lesions (Fig. 7) occurred in 14 cases (37%). Hypertrophic

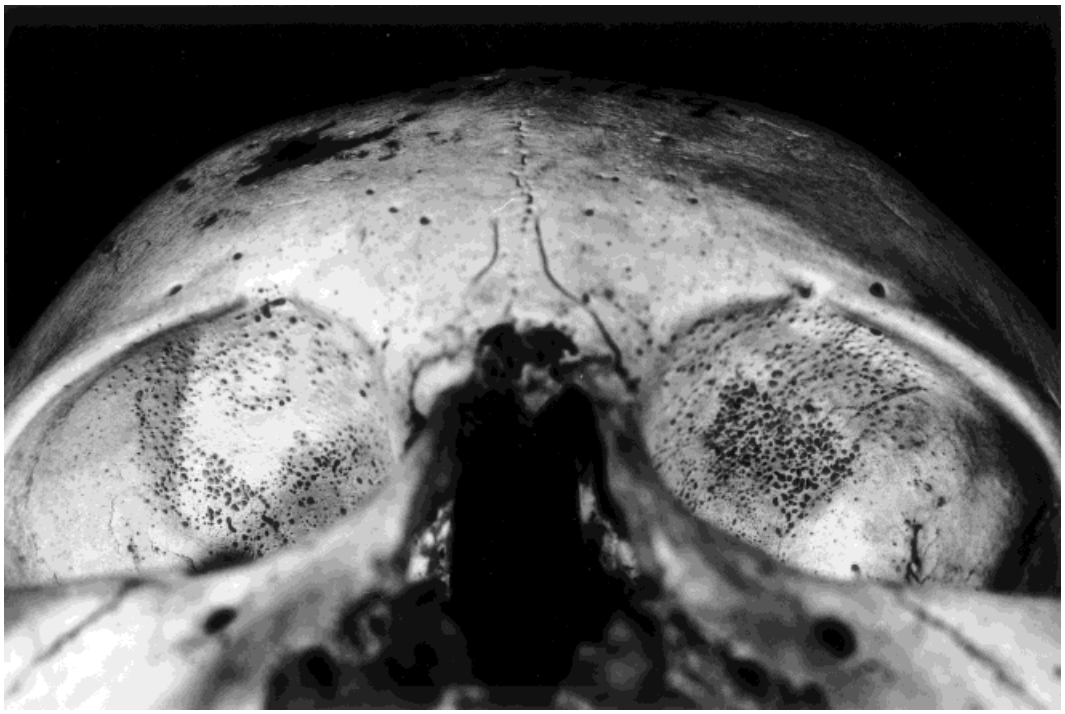

Fig. 5. Porous lesions of the orbit, frontal (roof) in NMNH 293169 from the site of Chilca, Peru.

TABLE 5. Frequency of other pathological features in skulls exhibiting abnormal cortical porosity of the greater wing of the sphenoid

	Absent	Present	Not observable
Greater wing of sphenoid hyperostosis	36	1	1
Cranial vault porosity	13	25	
Cranial vault hyperostosis	31	7	
Orbital roof porosity	1	37	
Orbital roof hyperostosis	15	23	
Orbit, lateral margin porosity[1]	24	14	
Maxilla, posterior porosity[1]	5	31	2
Zygomatic bone internal porosity[1]	6	30	2
Infraorbital foramen porosity	14	22	2
Infraorbital foramen hyperostosis	35	1	2
Palate porosity[1]	18	17	3
Coronoid process porosity[1]		3	35

[1] No cases of hyperostosis were associated with this anatomical site.

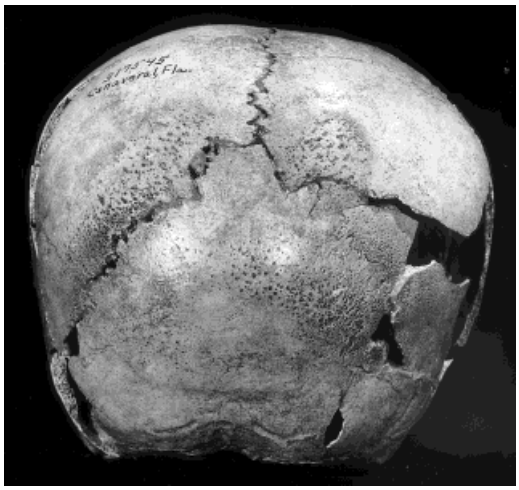

Fig. 6. Abnormal porosity of the lambdoid area of the skull in a child four years of age. NMNH 377545 from the site of Canaveral, Florida.

bone formation did not occur in the lateral orbit, the posterior maxilla, or the internal portion of the zygomatic bone.

Both the posterior surface of the maxilla and the internal surface of the zygomatic bone share, with the greater wing of the sphenoid, a close association with the temporalis muscle. Because of this, it is not surprising to find that the posterior maxilla (86%)

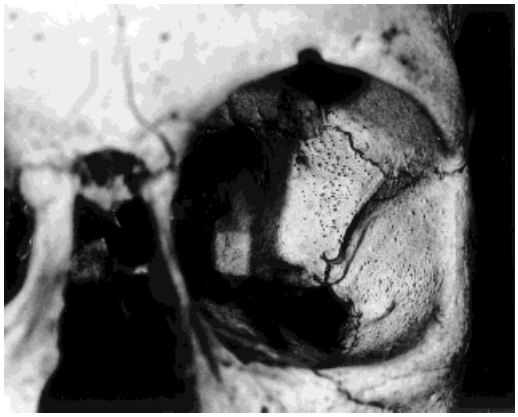

Fig. 7. Porous lesion of the left orbit, zygomatic bone (lateral) in NMNH 293169 from the site of Chilca, Peru.

Fig. 8. Porous lesions of the right posterior maxilla and internal zygomatic bone in NMNH 293169 from the site of Chilca, Peru.

and the internal zygomatic bone (83%) exhibit evidence of chronic inflammation with a high frequency of abnormal porosity (Fig. 8). There is little ambiguity about porosity on the internal surface of the zygomatic bone. This lack of ambiguity is not the case with the posterior maxilla because it also includes the area where the molars are erupting, and this is almost always associated with increased porosity in the associated alveolar process. For maxillary porosity to be defined as pathological, the area of porous involvement had to extend well beyond the alveolar process surrounding an erupting molar.

The infraorbital foramen provides a pathway for an artery, vein, and nerve supplying soft tissues that overlie the maxilla. Bleeding gums are a well-known clinical phenomenon in patients with scurvy, so abnormal porosity of the alveolar process should be associated with scurvy. The high frequency of porosity (61%) surrounding the infraorbital foramen in the scurvy cases was not anticipated. In one case, the porosity was accompanied by hypertrophic bone formation (Fig. 9), which may be indicative of a more chronic and severe inflammatory stimulus.

The greater palatine artery provides the vascular supply to the hard palate (Putz and Pabst, 1997) including the periosteum and overlying bone tissue. Ramifying vessels of this artery participate in the vascular response to chronic hemorrhage that occurs in the alveolar processes and the adjacent hard palate. It is this secondary vascular response that stimulates the increased porosity apparent in the bony palate of scorbutic subadults. Evaluating evidence of abnormal porosity of the palate poses problems that are analogous to those described for the posterior surfaces of the maxilla. In the normal palate, porosity is distributed in a u-shaped arc that roughly parallels the arch of the alveolar process. However, the porosity is denser in the anterior portion of the palate. As is the case in many pathological conditions, there is a fairly continuous gradient between the normal and abnormal, with the boundary being somewhat arbitrary in some cases. Often, however, the denser area of porosity, normally only in the anterior portion of the palate, extends markedly onto the posterior palate (Fig. 10). The increased density of porosity along with the abnormal posterior extension we define as pathological. Abnormal hard palate porosity occurred in almost half (49%) of the scurvy cases in this report and suggests that inflammation is a common feature in this anatomical area as well.

The temporalis muscle inserts primarily on the medial surface of the coronoid process of the mandible. Only three of the scorbutic subadult skulls in this report had associated mandibles, but all three showed evidence of abnormal porosity (Fig. 11) in the bone tissue associated with the insertion of the temporalis muscle.

Fig. 9. Porous and hypertrophic lesions of the suborbital foramina in NMNH 293169 from the site of Chilca, Peru.

DISCUSSION AND CONCLUSIONS

Nutritional diseases in archeological skeletal samples provide data on diet in earlier human populations. Scurvy has been reported in some archeological skeletal samples (e.g., Maat, 1982; Carli-Thiele, 1996). However, this nutritional deficiency is undoubtedly underrepresented in osteological studies of archeological human remains. The publication of the paper by Ortner and Ericksen (1997) on the anatomical features of scurvy in the skull provides the basis for new research on this important subject. The association of a pattern of skull lesions with scurvy is, of course, an inference based on careful integration of anatomical and pathological knowledge. Direct evidence of a link between the lesions reported in this report as well as that reported in Ortner and Ericksen (1997) and anatomical evidence of scurvy from cases of clinically documented scurvy has not yet been obtained. However, we argue that chronic bleeding at multiple sites in the skull is the most likely cause of the type and pattern of lesions reported in this study and that the only plausible diagnostic option for this particular type and pattern of lesions is scurvy. This report is the first attempt to explore the implications of this inference in a large sample of archeological human remains.

When this or any method is applied to the analysis of nutritional diseases in archeological human populations, it is crucial to fully appreciate the complexity of the problem. Malnutrition increases the risk of other diseases, such as infection. It is also the underlying factor in scurvy and rickets and a significant factor in iron deficiency anemia. Each of these diseases has somewhat different skeletal manifestations in the typical case where there is only one type of dietary deficiency. However, as we have noted in the introduction, each of these diseases

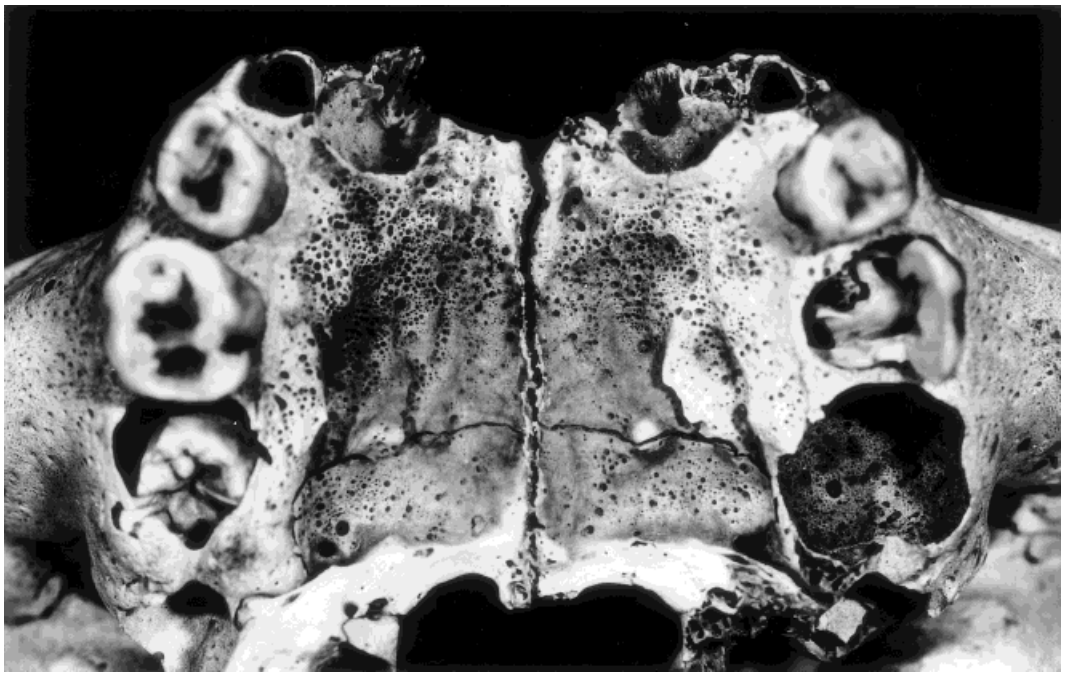

Fig. 10. Abnormal porosity of the maxilla, palatine process, and the palatine bone in NMNH 293169 from the site of Chilca, Peru.

Fig. 11. Abnormal porosity of the left, medial coronoid process in the mandible of a child 4 years of age. NMNH 377545 from the site of Canaveral, Florida.

can occur in any combination with any or all of the other nutritional diseases, and this can lead to challenging differential diagnosis even if the assumptions made about the link between a specific type of bone pathology and these diseases are correct.

In the Peru subadult skeletal sample, there is no convincing evidence of either bone deformity that could be associated with rickets or marrow hyperplasia that would support a diagnosis of anemia. For these reasons, it seems likely that scurvy is the only cause of the pattern of lesions described in this study. However, as part of continuing research on scurvy in North American archeological skeletal samples, the first author has seen a subadult skull from a site in Florida where the anatomical evidence argues strongly for both scurvy and anemia being present in the same subadult.

It is clear in Table 1 that scurvy was occurring in many areas of Peru, including coastal and highland sites. Very large samples would be needed to determine if there was any variation related to time or geographical location in Peru. The limited data presented in this study suggests that neither of these variables is associated with variation in the prevalence of scurvy.

The anatomical features we have presented in this study, particularly those seen in the orbital roof, could be confused with those occurring in anemia. Careful attention to the type of lesion and the distribution

patterns of the lesions is crucial in differential diagnosis. What is clear is that the tendency of paleopathologists to attribute porous lesions of the skull and particularly the orbital roof to anemia needs to be reevaluated. Diagnosis of anemia in the skeleton requires evidence of marrow hyperplasia. The results of this study strongly suggest that skulls with abnormal porosity but lacking evidence of marrow hyperplasia are more likely to be caused by other pathological conditions, such as scurvy.

We do not have completely satisfactory explanations for the porosity apparent in many sites of the skull. The most probable cause was some stimulus to increase vascularity. Bleeding is almost certainly the triggering condition for many and perhaps all the sites. The lesions apparent on the greater wing of the sphenoid are linked to the special anatomical features of the vascular supply to the temporalis muscle and the function of the muscle in chewing. The abrasion of weakened blood vessel walls in scorbutic children during chewing is a plausible explanation for the evidence of increased vascularity seen in the greater wing of the sphenoid. However, we see evidence of increased vascular supply at sites such as the orbital roof and the posterior maxilla where this explanation is less adequate. This suggests that abnormal bleeding may be caused by mechanisms other than abrasion. It seems likely that any mechanical strain, such as bending or stretching, on a defective blood vessel could cause the bleeding that results in the evidence of increased vascular supply through a cortical bone surface.

Further refinement of our understanding of the anatomical features associated with scurvy is needed. We have chosen a conservative strategy in diagnosing scurvy in the Peru subadult sample. With additional refinement of our understanding of skeletal changes in scurvy, the prevalence of scurvy reported in the Peru subadult sample would likely increase. One topic where research is needed is on the boundary between normal and abnormal porosity is in alveolar bone. Additional research is also needed on abnormal woven bone development on the greater wing of the sphenoid in infant skulls. It is also possible that other sites of inflammatory bone changes will be identified as more samples are analyzed. What is emerging is a new and important diagnostic tool in reconstructing one aspect of diet in archeological human populations.

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